Mid-systolic septal deceleration

A new sign of left ventricular outflow tract obstruction obtained by color-coded tissue Doppler echocardiography

Introduction

Hypertrophic cardiomyopathy is associated with a variety of clinical symptoms and hemodynamic alterations. A clear distinction has to be considered between the obstructive and the non-obstructive form for diagnostic and therapeutic reasons [6, 8, 14]. Basal septal hypertrophy, mitral valve malformations and malpositioning of the subvalvular mitral apparatus contribute to the development of dynamic....
obstruction of the left ventricular outflow tract (LVOT) [4, 8, 11]. The noninvasive diagnosis of hypertrophic obstructive cardiomyopathy (HOCM) is based on the identification of asymmetric septal hypertrophy with systolic anterior motion (SAM) and the presence of a systolic LVOT gradient as assessed by means of Doppler echocardiography [3, 7, 14]. Many patients present with no significant resting gradient, but develop significant obstruction after provocation like physical activity, induction of an extrasystole or pharmacologic intervention [10, 16, 17]. Doppler evaluation of the LVOT gradient during such maneuvers remains technically challenging and the interpretation may be complicated by the presence of concomitant mitral regurgitation. We describe a new echocardiographic sign for LVOT obstruction obtained by color-coded tissue Doppler echocardiography (TDI, Fig. 1), which is easy to record and which might help to identify affected patients and to monitor therapy.

Patient and methodology

A 69-year old woman with HOCM was referred for catheter-based treatment by transcoronary ablation of septal hypertrophy (TASH) using selective septal branch injection of ethanol [2, 12, 13, 21]. The diagnosis of typical subaortic HOCM had been made three months earlier by echocardiography on the basis of asymmetric left ventricular hypertrophy (enddiastolic septal diameter 19 mm, posterior wall 13 mm) and SAM. At rest, a low LVOT peak gradient of 24 mmHg (peak velocity 2.5 cm/s) was measured, which increased to 155 mmHg (peak velocity 6.2 cm/s) after bicycle exercise (50 Watts).

TASH was performed as described previously with an „over-the-wire“ technique [6, 13, 21]. Pressure recordings were performed continuously between a pigtail catheter at the apex of the left ventricle and the guiding catheter placed in the ostium of the left coronary artery to record the outflow tract gradient (peak-to-peak) at rest and after provocation by stimulated premature ventricular beats at a fixed coupling interval of 400 ms.

Simultaneous with the TASH procedure, we performed serial transthoracic echocardiographic examinations from the apical four-chamber view (Vivid 7, GE/Vingmed, Horten, Norway). The LVOT gradient was measured by continuous-wave (CW) Doppler and longitudinal septal motion was recorded by color-coded TDI (>96 frames/second) and digitally stored for post-processing (Echo Pac PC, v. 3.0.0, GE/Vingmed, Horten, Norway). Previous phantom testing documented no significant temporal delay between the recorded ECG signals and the CW-Doppler spectrum and the color-coded TDI velocity trace.

There was only a small LVOT peak-to-peak pressure gradient of 30 mmHg at rest (Fig. 2a, top). The corresponding tissue Doppler velocity trace was characterized by an early systolic peak, followed by a sharp deceleration and a late systolic plateau (Fig. 2a, middle). The stimulated post-extrasystolic peak-to-peak LVOT gradient was 191 mmHg by invasive pressure recording (Fig. 2b, top) and >110 mmHg by Doppler (largest recorded Doppler velocity 5.3 m/s, Fig. 2b, bottom). The simultaneously recorded TDI velocity trace showed an increased peak systolic velocity with a marked systolic deceleration notch (Figure 2b, middle). Five minutes after injection of 1.2 ml ethanol into the first septal branch of the left anterior descending artery, a small post-extrasystolic peak-to-peak gradient of 20 mmHg was documented by invasive pressures and Doppler (Fig. 2c, top & bottom). The corresponding post-extrasystolic TDI image showed a lower peak systolic velocity and less deceleration than at baseline before ethanol injection (Fig. 2c, middle).